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A CASE OF ANOMALOUS CENTRAL RETINAL BLOOD VESSELS—ATROPHY OF THE OPTIC NERVES.

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Mr. A. M., from the State of Illinois, aged 56, consulted me on October 9, 1884, on account of impaired vision.

He is a farmer by occupation, has served during the war and acquired the alcoholic habit to which he has been addicted ever since. He does not recollect of having had a disease of any kind. The family record is apparently good with the exception of that of his aged mother who has been blind for many years from a cause unknown to the patient.

Status praesens.—Patient is tall, well developed. Face puffed, cheeks and nose striated with dilated capillaries. Lower lids and conjunctiva bulbi slightly œdematous. Scleral veins enlarged and somewhat tortuous. Irides are grey, showing perfectly the outer and inner circles with the intermediate crypts and trabeculæ. Anterior chambers narrow. Pupillary margins somewhat jagged, but no posterior synechiæ nor are there pigmented deposits on the lens capsule as remains of former adhesions. The pupils are greyish, myotic and react slowly upon light.

By oblique illumination it is seen that both corneæ are clear, no opacities are found in the aqueous humor, and no pathological changes on the anterior capsule or in the immediately adjacent portions of the lens.

In examining the lens-system by means of reflected images, I find the corneal and the posterior capsular images normal, but I am unable to discover the anterior capsular image. This is, probably, due to the myotic condition of the pupil which brings the three images into close proximity and thereby causes a suppression of the anterior capsular image by the powerful corneal reflex. The posterior capsular image is clearly discernible in the entire pupillary field.

Upon dilating the pupil *ad maximum*, the fundus is seen perfectly clear and no traces of opacities behind the lens are to be found.

Examining the optic nerve O. D. in the direct method with Landolt's ophthalmoscope, using a plane mirror, I considered its size normal, possibly somewhat diminished. The shape of the optic papilla is round, it is bordered with an accumulation of dark brown pigment at the upper and inner quadrant. The scleral ring is clearly visible in its entire circumference. The peripheral portions of the optic papilla show a very faint reddish color to the width of a semi-radius; this color gradually increases in intensity towards the center and it ends in an abrupt margin at the middle portion of the optic nerve, which is excavated, intensely white and studded with bluish well marked irregular spots.

The central blood vessels pass from the retina over the scleral ring into the peripheral portions of the optic papilla in the same plane, stopping abrupily at the margins of the excavations; their color becomes intensified and they change their direction somewhat and gradually disappear through the lamina cribrosa. Arteries as well as veins are somewhat tortuous; the former are reduced to two-thirds of their normal caliber. In regard to the origin and distribution of the blood vessels there is nothing particular to relate excepting the following, viz: that the vena temporalis inferior instead of being distributed over the inferior temporal region of the retina, forms a ramus recurrens. Com-

ing out of the lamina cribrosa with the other blood vessels, it follows about its usual course up to the margin of the excavation, when after having made an abrupt bend outwards, it runs almost midways between the margin of the excavation and the periphery of the optic papilla and very nearly parallel with the scleral ring. (See figure 1.)

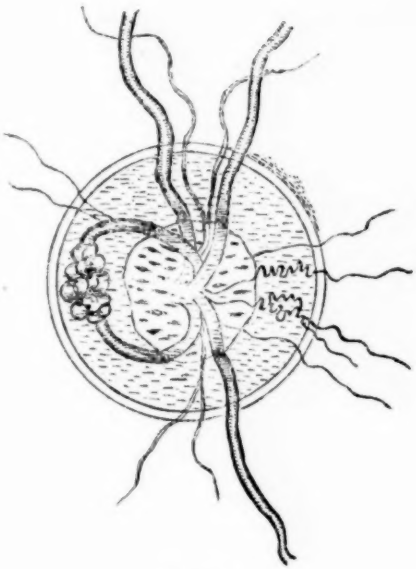

Figure 1.

After having traveled upon the disc over a space of about one-fourth of its diameter, the vein forms a cluster of tortuosities, all of which combined assume the shape of a mulberry of about one-fifth of the size of the disc lying close to the peripheral portion of the optic papilla. The contours of the whole and of each individual convolution are exceedingly plane, showing in every convolution a central reflex and two dark red lines on each side as is characteristic of an ordinary blood vessel.

This feature, however, is planest upon the more superficial ones. Pulsation is marked, but I am unable to say, whether the wave of pulsation, starting from the cluster, went towards the

center by way of the vena temporalis inferior or by the superior recurrent portion.

The recurrent portion passes inwards in a curve until it reaches the temporal margin of the vena temporalis superior, then passing over it to the margin of the excavation makes a sharp bend towards the center of the papilla and is lost in the physiological excavation between the vena temporalis superior and the vena nasalis superior.

On the nasal side of the disc, two small veins are seen, which also show very marked tortuosities while passing over the peripheral portion of the papilla. The lower one gives off a branch which behaves in the same manner. V=fingers at $2\frac{1}{2}$ metres.

The peripheral portions of the optic papilla lie nearly in one plane with the inner surface of the surrounding retina. However, the central light portion of the optic disc, to the extent of about one-half of it, is as stated very much excavated.

Correcting my own myopia by a—4 D lens, I am able to see the fundus of the excavation with its vessels and nerve bundles distinctly. Next, observing the non-excavated portion of the disc and the adjacent retina, both of which, as I have stated above, were practically in the same plane, I find that—2.50 D corrects the refraction. In looking at the summit of the cluster of blood vessels on the optic papilla—1.75 D gives me a clear picture. Thus I may derive the following conclusions, viz., that the distance from the anterior pole of the cornea to the most excavated portion of the optic papilla is equal, or very nearly so, to the optical axis of an emmetropic eye, that the fundus of the eye and the peripheral portion of the optic nerve present a hyperopia (or shortening) of +1.50 D, or $\frac{1}{2^{\cdot}4}$ old series, and furthermore that, in order to see the cluster of vessels distinctly, an additional lens of +0.75 D was necessary, which showed a second shortening.

Having found hyperopia 1.50 D or $\frac{1}{2^{\cdot}4}$ old series as the refractive condition of the non-excavated portion of the optic papilla and knowing also the refractive condition of its excavated portion and of the surface of the cluster of blood vessels, it is an easy matter to compute their distances from each other, referring

to H. Knapp's "Table, indicating the ophthalmoscopic determination of the shortening, resp. elongation, of the ocular axis by means of positive, resp. negative, auxillary lenses," published in Knapp-Moos' Archives of Ophthalmology and Otology, Vol. III·, No. 2, Page 19.

The non-excavated part of the optic papilla compared with the fundus of the excavation which shows H 1.50 D or $\frac{1}{24}$, the ocular axis must be shortened 0.467 mllm., or, what is essentially the same thing, the excavation of the optic papilla must be 0.467 mllm., deep. The height of the cluster of blood vessels being seen by an additional +0.75 D or $\frac{1}{48}$ lens, this shows a shortening of 0.322 mllm. of the ocular axis, or in other words, the cluster of vessels is 0.232 mllm. high.

Perimetric examination reveals a concentric diminntion of the field of vision, but no scotoma.

The other eye shows a physiological evcavation with atrophy of the optic nerve.

CASE OF RETINAL HÆMORRHAGE APPARENTLY DUE TO SIMPLE ANÆMIA, WITH REMARKS.

BY J. F. FULTON, M. D., PH. D.,

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Although cases of retinal hæmorrhage are frequently met with as the result of various obstructive troubles of the circulation and serious diseases giving rise to organic disorders of the walls of the blood vessels, and of pernicious anæmia. All authorities agree that it is a very rare result of simple anæmia; so scarce indeed that but few well authenticated cases have been reported and with some of these there has been the suspicion that the hæmorrhage might have been due to other causes. It is this interesting factor in ætiology of retinal hæmorrhage together with a perfect recovery of sight after a severe effusion of blood at the yellow spot which renders the case worthy of publication.

Miss R., aged 26, was sent to me by Dr. Briggs of this city. She was somewhat feeble, but a careful examination of her physical condition revealed no other trouble than that of simple anæmia. And the cause of the anæmia was not apparent as she never had been the victim of any profuse loss of blood; her digestion was good, although appetite rather captitious. There was no history of malaria or of any constitutional disorder, but the blood was watery and deficient in red corpuscles. The other indications of anæmia were the palor of her lips and tongue, the weak and somewhat accelerated pulse, the rapid production of fatigue by exercise. But she did not complain much and thought her general health quite good. There was no murmur over the heart nor anything to indicate disease of that organ, except slight palpitation after excitement or upon exertion. A few days before coming to me for advice, just as she was coming out of a cold bath she noticed that a large scotoma showed itself suddenly before the right eye and that she could not see anything distinctly with that eye. At the time of the examination the acuteness of vision in the eye was $\frac{20}{CC}$; of left eye $\frac{20}{XX}$. The ophthalmoscopic examination showed that both eyes were emmetropic and that the background of the left eye was perfectly healthy, but it revealed a large fan-shaped hæmorrhage of the right eye, entirely obscuring the macula lutea and extending towards the optic disc. It was dark purplish in color. There was but the one hæmorrhage. The disc and retina were pale, and the blood vessels rather more transparent than normal.

She complained much of the annoyance which the scotoma gave her, but insisted that her sight was perfect before this accident happened.

As the above notes indicate, no other cause could be found to account for this young lady's trouble than that of simple anæmia, and it was with that idea in view that she was treated. She was put upon full doses of iron and a rich and liberal diet, under which she rapidly improved. We were gratified to notice that the hæmorrhage became thinner from time to time and the acuteness of vision slowly but surely improved, and that the scotoma changed its color and became "thinner" from day to day. The clot of blood became decolorized centripetally and

could be noticed becoming less and less dense at each successive ophthalmoscopic examination.

Hæmorrhages in the region of the yellow spot are worthy of our most careful study, on account of their frequent occurrence and very great importance; as they not infrequently produce a permanent defect in the acuteness of vision. In such cases, however, it is likely that the effusion takes place into the deeper layers of the retina and results in a disorganization of the tissue and consequently a loss of function. In those cases, like the one reported, in which there is a complete recovery of sight and no permanent lesion remains at the seat of the hæmorrhage, we must assume that the blood lay between the vitreous body and retina, or upon the inner surface of this tunic.

The ophthalmoscopic appearance of the apopletic spot gave this impression as the retinal blood vessels could not be seen behind the clot, but, had the hæmorrhage taken place into the choroid, these blood vessels would not have been hidden from view.

Wolfe, in referring to this subject in his work, makes the following practical conclusion: "That ophthalmoscopic appearance does not always coincide with the extent of the retinal lesion. Thus, sometimes, we are surprised to meet with a case of complete abolition of sight while the ophthalmoscope shows only a serous transudation; and, on the other hand, in cases where vision is very slightly affected, we notice extensive apoplectic patches within retinal sclerosis." I am inclined to think that a more careful study of the relation of the blood clot to the retina as to depth will assist us greatly in giving a more correct prognosis. None of the usual causes of retinal hæmorrhage was present in this case. There was no history of injury, no derangement of the vascular system, none of the graver alterations of the blood, such as albuminuria, hæmorrhagic diathesis, diabetes, or leucocythæmia, etc. There was nothing to indicate inflammatory change of any kind. Dr. Stephen Mackenzie in a paper read before the Ophthalmological Society of the United Kingdom narrated a series of carefully studied cases which seemed to indicate that a tendency to retinal hæmorrhage was produced wherever the corpuscular richness of the blood

fell below 50 per cent, thus indicating that anæmia is one of the causes of retinal apoplexy.

Dr. Henry Juler in the *Ophthalmic Review* quotes Dr. Gowers as saying that in simple chronic anæmia retinal hæmorrhages are "very rare, and probably only take place where there is a great absolute deficiency in the number of red corpuscles."

Although no estimate of the percentage of red blood corpuscles was made in the case I have here reported, yet judging from the facts that the patient was anæmic, that all other causes of retinal hæmorrhage were absent, that the patient improved rapidly under large doses of iron, I think that this can be classed without hesitation as one of retinal effusion of blood, the result of simple anæmia.

MISCELLANEA FROM PRACTICE.

ADOLF ALT, M. D.

I.

An Unusual Case of Atropine Poisoning.

On October 16, 1879, Miss J. McF., æt. 39, consulted me on account of floating shadows, light-flashes, and gradually increasing dimness of vision in both eyes. These symptoms had become more aggravated for about six months previous to her visit, and she dated their origin from the 6th of April, the same year, on which day she had taken a severe cold. Her eyes got inflamed at the same time, were red and painful, and remained so for a prolonged period. At the same time she had, moreover, an eruption of the skin all over her body. (When consulting me, patient was engaged to be married to a clergyman, and wanted her sight restored before the marriage, which was set for a not distant day.) Although everything pointed to a specific disease, I could not elicit anything further from her, and the physician who had sent her to me considered such a thing as absolutely impossible, and my diagnosis as absurd.

Status præsens.—Outward appearance of the eyes normal. Anterior chamber O.U. shallow, pupils small. V. in R.E. $=\frac{20}{LXX}$, in

L.E. $\frac{20}{cc}$ difficult. R.E. reads Jæger 8 with difficulty. After the instillation of atropia there appeared in the L.E. a posterior synechia of the iris downwards. The periphery of the lens was dim, striated. The optic disc was swollen and whitish infiltrated, its outlines invisible; the retinal veins were tortuous, but arteries and veins as far as seen appeared very small, owing to a thin exudation that apparently covered them. Floating opacities. In the R. E. the vitreous was generally dim. Optic papilla and retina were in the same condition as in L.E. Glands of the neck swollen and hard.

I felt satisfied that the former iritis, as well as the optic neuritis now, was a specific affection, and I told the lady, without mentioning the real cause, that she would have to undergo a vigorous treatment for a constitutional ailment, of which her eye affection was but a symptom. The physician who had brought her to me insisted, as I said, upon my being mistaken in the diagnosis; and I heard nothing further of the case until March 13th of the next year, when she unconditionally accepted vigorous anti-syphilitic treatment, which consisted of mercurial inunctions, iodide of potassium in half-drachm doses three times a day, leeches to the temples, rest in bed and a dark room.

Under this treatment the vitreous body cleared up in both eyes, and on the 27th of that month the outlines of the optic papillæ were plainly visible. On the 30th the fundus of the L.E. was apparently normal, while in the R.E. there was yet a slight haziness, and the retinal veins were as yet tortuous.

Thus everything was going on well, when on April 1st I was sent for at 10 P. M., on account of very alarming symptoms, which had set in during the forenoon. I found her slightly delirious, the face hot and flushed, skin dry. The pulse was 92 and fluttering, irregular. She complained of severe headache and dizziness, a dry throat and heavy tongue, and spoke very slowly. Her limbs were numb, and her menses had appeared before their time. Her pupils were dilated *ad maximum*, the fundus of both eyes appeared exactly as it had done two days before.

She had not had any atropine or belladonna by my order, either internally or instilled into the conjunctival sac; but she had

had a bottle with iodide of potassium refilled the preceding day, and taken several doses of this medicine.

The question was now whether all these symptoms were due to anatomical changes in the cranial cavity (gumma, malignant tumor, etc.,) or not. The condition of the optic nerves in both eyes being absolutely unchanged, I did not think it probable that an intra-cranial affection had caused these alarming symptoms. The next probable thing then was that she was poisoned by belladonna or atropia. Two things, however, spoke against this diagnosis, viz: The comparatively slow pulse, and the fact that no belladonna in any form had been prescribed.

I, therefore, being somewhat in the dark, gave her simply a subcutaneous injection of morphine, had an icebag applied to her head, and discontinued all other medication. The bottle I quietly put into my pocket.

The next morning I found patient was considerably improved. She had vomited several times during the night, and had fallen asleep at 7 A. M. Her face was no longer flushed, she talked rationally, though yet slowly and with a heavy tongue. Pulse regular, 60. Pupils the same as before. The numbness of the limbs was gone; she only complained of a "globus" in her throat.

April 3d.—All the symptoms have nearly disappeared. The pupils are beginning to contract. Fundus U.O unaltered.

April 10th.—V. is $\frac{20}{LXX}$ O.U., reads Jæger 5 with difficulty. The fundus in both eyes appeared normal.

I had the iodide of potassium solution examined by a reliable chemist, who made his analysis without knowing of the case in consideration. He reported that the solution contained atropia and iodide of potassium.

II.

A CASE OF POISONING BY DUBOISIA

Mr. E. M. D., æt. 32, came under my treatment on account of a severe non-specific iritis of the right eye.

He was put to bed in a dark room, atropine was instilled and leeches were applied. Whenever pain came on some instillations of a four per cent cocaine solution were made and they relieved him

usually. His pupil not being fully dilated after several days of atropine instillations (about 1 per cent.) every half hour, and because his conjunctiva seemed to show some atropine granules, I changed the mydriatic into duboisinum sulfuricum of the same strength, to be instilled every hour.

This solution the patient received in the evening at nine o'clock, and he used *three* drops of it, according to his own statement and actual measure.

At midnight the nurse noticed that he was talking wildly, looked flushed, and tossed about in his bed, and, therefore, removed all his medicines from him. In the morning he jumped out of bed and tried to run off, but fell down powerless. His brother who had accidentally dropped into the hospital to see how he was doing, came running to me, perfectly satisfied that his brother had gone crazy. Not being able to go at once and see the patient myself, I sent the brother back to the hospital to have the patient given morph. sulf., gr. $\frac{1}{8}$, and chloral. hydr. grs. 15, every half hour. This was at 11 A. M. I also send for a colleague to see the patient with me in consultation, but did not succeed in doing so.

At 2 P. M. I saw the patient myself and found him in a slight slumber. His face was yet flushed, his pulse 136, skin dry. He complained of very severe headache, dry throat and mouth, and told me that he had to talk nonsense, although he knew it was such and tried hard to help it, and he begged of me to help him over this fearful condition.

For a moment I thought I would gvie a him large quantity of muriate of pilocarpine hypodermatically, but seeing that five doses of morphine and hydrate of chloral of the strength above mentioned had actually quieted him and seemed well to counteract the poisonous effect, I concluded to keep on giving these anodynes at longer intervals.

When I saw him in the evening he had had a short sleep; but his pulse was yet 112 and he yet talked delirious. The nurse said, he was getting more restless again. I therefore ordered her to push the morphine and hydrate of chloral with precaution, until the patient should fall soundly asleep. This was finally succeeded in after the patient had again taken six-eighths of a grain

of morphinum sulfuricum and one and a half drachms of hydrate of chloral.

He slept from midnight until morning without waking and then dozed nearly all day, but was easily aroused. His pulse was 84 and the dryness of the skin had given way to a slight moisture. All symptoms of poisoning gradually disappeared.

As the solution of duboisinum sulfuricum contained gr. 2 to ℥ss of water and he had used of this solution just 3 drops, he had been poisoned by three doses of gr. $\frac{1}{120}$ or gr. $\frac{1}{40}$ in all, and it took not quite gr. 2 of morphine and ℥½ of hydrate of chloral to get him into a safe condition.

The second day I began again to have atropine (gr. 4 to ℥1) instilled and no further disagreeable symptoms occurred.

III.

A Case of Keratitis Phlyctænulosa of Long Standing, Healed after Enucleation of the Phthisical Fellow Eye.

W. H., 8 years of age, was brought to me July 27, 1879, with a history of phlyctænular keratitis which had then existed for four months in the L.E., and for something over three months in the R.E. There was swelling of the lids, lachrymation, pain, photophobia, and blepharospasmus. As it was impossible to examine the eyes without an anæsthetic, I put the boy under the influence of chloroform.

Status praesens.—R.E.: Considerable injection of the conjunctival vessels, two phlyctænulæ near corneoscleral margin. Large central ulcer. L.E.: Cornea very vascular. Large perforation lying somewhat eccentrically, iris protruding through it. Margins of ulcer infiltrated with pus. Protruded iris yellowish discolored. No anterior chamber. T=—2.

I removed the prolapse at once with the scissors, and put the boy under the usual treatment of cold applications, yellow oxide of mercury ointment and atropia. The ulcers in both eyes healed rapidly. In the L.E. the basis of the healing ulcer, however, gave way several times again to the increasing intraocular pressure. No further protrusion of the iris, however, occurred.

August 10th.—The anterior chamber in L.E. refilled. Both eyes very irritable. New phlyctænulæ in R.E.

This condition remained about the same for a short time. Then the L.E. began slowly to lose its vision. The R.E. got alternately better and worse.

September 9th.—L.E.: Visual field concentrically constricted. T=—3. Adherent leucoma, beginning phthisis anterior.

As the phthisis of the L.E. progressed it became totally blind,

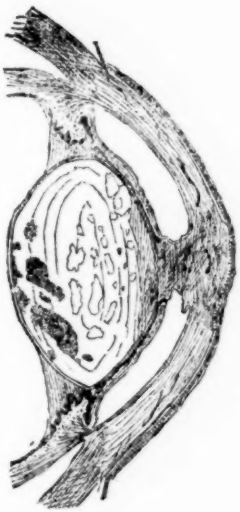

Figure 2.

and remained irritable and tender to the touch. The condition of the R.E. did not alter, in spite of the treatment.

A month afterwards I therefore concluded to remove the L.E, After this was done the photophobia of the R.E. and the formation of phlyctænulæ ceased at once. The boy had no relapse during the year following. Since then I have not heard of the case.

The clinical aspect of this case has many features in common with the cases which have by several authors been described as cases of sympathetic keratitis, and the result of the operation is

such that it looks as if we had in such cases really to deal with a sympathetic affection of the cornea, or at least that a shrunken and chronically inflamed eye can keep up an existing affection of the fellow by sympathetic influences.

The removed eyeball had several interesting features. I had thought that the retina must be found detached to its whole extent. But to my astonishment the macroscopical specimen showed no detachment of this membrane. Under the microscope, however, there were found innumerable—so to speak—microscopical detachments of the retina, caused by the exudation of (if I may so express it) small drops of a gelatinous material between choroid and retina. This exudation was found to lie either between the pigmented epithelium and the choroid, or between the pigmented epithelium and the retina.

A further uncommon feature, which is shown by figure 2, was that a considerable amount of blood was found within the lens-capsule. This membrane had probably been ruptured at the time when the perforation of the cornea had occurred. The opening was, however, so small that I could only find it with great difficulty. The infiltration of the lens-substance with blood had apparently taken place, also, at the same time, when the rupture of the lens-capsule had occurred since no hæmmorrhage was found in what existed still of the vitreous body. Otherwise the lens-substance appeared unaltered.

IV.

Cataract Extraction Followed by Death.

A case of death after the extraction of a cataract is certainly a rare occurrence, and, as I know of only one further case, communicated to me in correspondence with one of the leading operators, I think it may be well to put my unfortunate experience on record.

On October 14, 1880, Mr. W. C., ætat. 80, consulted me on account of loss of vision in both eyes, which had been gradually coming on during the last four years.

I found the gentleman to be healthy and strong for his age, and walking quite firmly. In both eyes I found a mature nuclear cataract. Pupils dilated well upon instillation of atropia. Ten-

sion, as well as function of the eyes normal. I advised extraction of one of the cataracts, and was called upon to perform this operation on October 18th.

I made a large corneo-scleral section and a large iridectomy. When the latter was completed, some bleeding into the anterior chamber took place. After the lens-capsule was opened I attempted the expulsion of the lens, but found that the corneo-scleral wound, though larger than average incision, was not sufficiently large to admit of the passage of the immense, hard and unelastic cataract. I therefore enlarged the wound with strabismus scissors, and attempted again to expel the cataract, when the patient gave a sudden jerk, and the cataract became dislocated upwards and backwards. After two or three attempts at reposition of the lens into the patellary fossa with a sharp hook, I succeeded in doing so, and then extracted the lens with the spoon with but a very slight loss of vitreous body. During the last steps of the operation the wound-lips were considerably bruised. After a few minutes of rest the patient counted fingers and could tell the time by the watch.

October 19th.—Had a tolerable night; slight pain; no undue irritation. 8 P. M.—Some mucoid discharge; no pain. A small bead of vitreous body in the corneal wound; cornea hazy; wound lips slightly infiltrated; no anterior chamber; some cortical substance in pupillary space; iris discolored.

October 20th.—Had a good night; no pain; no swelling; some muco-purulent discharge on the cotton; no chemosis; infiltration of the wound-lips more marked on the temporal side; shows well defined margins. 8 P. M.—Wound closed; some anterior chamber; some œdema of the conjunctiva; no pain; less discharge.

October 21st.—Had a good night; very little discharge; œdema of the conjunctiva; there is some necrotic material lying in the outer angle of the wound; anterior chamber larger; pupil not contracted; pupillary space clearer.

October 22d.—Had a very bad night on account of "weak spells" and atropia excitement (?); no discharge; no pain; less infiltration. Champagne.

October 23d.—Cornea clearing; wound-lips nearly free from infiltration; no discharge; pupil large, filled with cortical substance.

October 24th.—Wound clean; the patient counts fingers by candle-light close by; he eats a good meal with relish. To my surprise I found him at 9 P. M. exceedingly weak; tongue heavy; talks perfectly rational. Champagne.

October 25th.—Patient is very weak, and lies in a stupor, from which he is aroused with difficulty; he takes no food. Consultation with Dr. A. J. Temple; brandy enemata and ammonia.

October 26th.—8 A. M.—Patient has been slowly sinking during the night; œdema of lungs. 9 A. M.—Death.

The patient had never any fever; septic infection is, therefore, to be excluded. What caused his death, and whether his death was directly due to the cataract extraction, is a question I cannot answer. How dark the case remained to the consulting physician was shown by his certifying that death had been the result of "*secondary collapse*," whatever that may mean.

TRANSLATION.

The following is a translation of parts of a paper on movements of the cones and pigmented epithelial cells of the retina under the influence of light and the nervous system, by Th. W. Engelmann at Utrecht, which appeared in the *Archiv f. die Gesammte Physiologie*, etc.

* * * The inner segments of the cones become shorter when influenced by light and become elongated in the dark.

When the inner part contains a so-called ellipsoid, this does not, or at least comparatively little, change in form. The same is the case with the outer segments of the cones and with the rods. It seems that only that part of the inner segment of the cones is actively movable, which most resembles protoplasma and which lies between the membrana limitans externa and the outer segment. In this movement the moving part remains always in continuity with its cell-body in the outer granular layer. When it becomes shortened it gains in thickness, when it is elongated it becomes thinner in such a manner that probably no change

in volume takes place. In this it corresponds with contractile protoplasma or muscular fibres.

The absolute and the relative amount of the change in length of the cones differ in the various animals which have been examined, and they may differ very materially in the cones of one and the same eye and under the same conditions. * * *

* * * The rapidity of the movement is such that in frogs (which have been kept in the dark), the cones previously stretched *ad maximum* may be contracted *ad minimum* after having being exposed to diffuse daylight for several minutes only. If the animal is exposed to *direct* sunlight less time is required.

It seems that the elongation after sudden darkening generally needs a longer time than the contraction. It has not yet been possible to measure the time exactly. But, from what has been reported with regard to the frog, it is evident that the rapidity is of the same order as that of the movement of many forms of contractile protoplasma, for instance, that of the pigmented cells of the skin, of the contractile cells of the corneal epithelium, and especially of the pigment granules in the protoplasmatic offsets of the pigmented cells of the retina in the same animal.

Since the pigmented cells of the retina under the influence of the same light move in general to the same degree and in the same direction as do the cones, we might suppose, that both movements were due to the same cause, in such a manner that one could not occur without the other. There are, however, conditions in which the cones may be contracted *ad maximum*, while the pigment granules do not change the position they were in while in the dark, and vice versa.

It seems that all parts of the *visible* spectrum can produce this photo-mechanical reaction of the cones, if their action has only been sufficiently long and strong. * * *

* * * We have not yet examined whether the *invisible* (infra-red and ultra-violet) rays can call forth the same reaction. * * *

* * * We can conclude, that the place of the primary irritation lies inwards from the division between the outer and inner segment of the cones. Further experiments have made

it even very probable, that this place lies within the inner segments themselves, or better, in their contractile protoplasmatic substance. * * *

* * * These facts prove that the movements of the cones and of the pigmented cells of the retina are directly dependent upon the nervous system.

The author's attention was drawn to the possible existence of such a dependence by the fact, that when one eye only of a frog, previously kept in the dark, was exposed to light, the cones and pigmented cells were found in the position of maximum contraction near the limitans externa in the retina of the eye not so exposed. The only difference between the two was that the outer segments of the rods in the exposed eye were perfectly bleached, while in the unexposed eye they were as intensely tinted as if the frog had not at all been exposed to the light.

By repeating this experiment it was found that this photomechanical reaction of the pigmented cells and cones takes place in equal strength and at the same time in both eyes, when one only is exposed to light, and that when shielded from the light both resume their former positions *pari passu*. This was the same in bloodless, even in decapitated, frogs, when the brain was uninjured, at least a short time after the bleeding from decapitation. Later on the cones assumed more and more the contracted form in a similar way as do other contractile organs during "spontaneous" death.

When the brain had been destroyed by means of a knife or needle the influence of the light affected only the exposed eye. We can, therefore, exclude a direct aktinic irritation of the unexposed eye by light which might reach it from the exposed eye. * * *

* * * On the contrary, we are forced to assume an association of the cones and pigmented cells of both eyes by way of the nerves, *i. e.*, a "sympathetic" co-action of the two retinæ.

According to our present anatomical knowledge such an association can only be brought about by the optic nerves. Therefore these nerves act not only as centripetal nerves which conduct the sensation of light to the brain, but also centrifugally as motor nerves for the cones and pigmented cells of the retina.

It is not probable, that the sensitive as well as the motor impulses travel by way of the same nervous fibrillæ. * * * * * * Since this association of the two retinæ exists not alone in the frog but also in the pigeon (the only animal so far examined), it is undoubtedly of a very general occurrence among the vertebrates and is probably not wanting in man. It is then not impossible that in the latter the fibres of the anterior commissure of the chiasma of the optic nerves (the physiology of which has so far not been understood) have the function of accessory conductors. It is certain at least, that (in the *rana esculenta and temporaria*) retino-motor fibres go by way of the optic nerve from the large nerve-centres to the eyeball.

This conclusion was reached by the author, when he tried * * * whether it was possible to produce visible changes in the retina by throwing the light exclusively upon the skin of the body. The first experiment in this direction at once gave a positive result.

The heart and trunk of a frog, having been previously kept in the dark, while in the dark were put into an absolutely undiaphanous cap, made of four layers of thickest black velvet, which could be fastened on him in the manner of a tobacco pouch, and; to be absolutely sure, this again was put into a black, thick carton. Then the back and posterior extremities, while under a stream of water, were exposed for a quarter of an hour to the influence of sunlight.

The eyes which were at once enucleated in the dark and hardened, showed cones and pigment contracted *ad maximum* towards the limitans externa, as if they had been struck directly by the light. Of course, the outer segments of the rod were intensively tinted.

A frog taken from the same box, treated in the same way, but *not* exposed to light, showed in both eyes the common condition of a retina which has been kept in the dark. * * * * * * One thing is certain, that it is possible to causea reflex movement in the cones and pigment epithelial cells of the retina by the irritation of distant regions of the body. * * * * * * On the other hand, the author had occasion to convince himself of the fact, that it is not the light which is abso-

lutely necessary for the production of such movements. The retinæ of frogs, kept in the dark, made tetanic by strychnia and killed, showed the same position of cones and pigment, as do retinæ which have been exposed to light. The same effect was produced when the eyes of frogs kept in the dark were tetanized, *in vivo*, or at once after extirpation in a dark room, by moderate induction currents. Curare did not diminish the reaction, nor did it produce it.

In the same journal H. Aubert in a paper entitled: Does the Curvature of the Cornea Most Resemble an Ellipse? comes to the following conclusions:

* * * Is the cornea curved symmetrically, or does its curvature change at intervals? We must answer in the affirmative with regard to the nasal side, and state, that the cornea towards the nose becomes very rapidly flattened, whilst about 12 degrees to both sides from the optical axis the curvature is about equal. We are, therefore, forced by these observations to acknowledge two zones of different curvature, a marginal zone and a polar zone. The flat marginal zone goes over into the nearly symmetrically curved polar zone with a rapid increase in curvature.

Thus we can distinguish between two zones in the cornea, of which one, the marginal zone, remains in accordance with the anatomical conditions of the eye-ball, while the other, the polar zone, serves the optical requirements. The form of the eye-ball is such that the part defined by the sclerotic is almost spherical with a radius of from 11 to 12 mllm.; the margin of the cornea having almost the same curvature as the sclerotic is a direct continuation of the sclerotic. This part then passes over gradually into a part of the cornea of a stronger curvature in a zone of the cornea which is immaterial for optical purposes, since the rays falling upon it are kept from entering the eye-ball by the iris. It is only where this sclerotic curvature of the bulbus is totally changed, that the part of the cornea begins which is material for vision and which lies in an area of from 12 to 16 degrees around the optical axis. The curvature of this part is such that by it the rays of light are refracted in such a manner as to unite upon the retina. We may, there-

fore, call the polar zone of the cornea its *optical* zone, and the marginal zone its *sceral* zone. Their limits are given by the iris and pupil.

D. Waldhauer in an article on four cases of diabetic cataract published in the *Revue Générale D'Ophthalmologie* makes the following remarks:

O. Becker in his book on the crystalline lens has given more consideration to the diabetic cataract than any of the writers on the same subject. He states, and I can confirm it from the seven eyes I operated upon, that the opacity lies with preference in the cortical substance and that the nucleus is comparatively transparent.

I have also been struck by the ease with which it is separated from the cortical substance. The cataracts had very quickly become ripe and in two cases regressive metamorphosis had rapidly taken place. I have not seen in my cases any particular swelling of the cataract, nor the changes in the pigment of the iris, which are described by O. Becker and Hirschberg.

The results of the operations in the four patients may be considered very favorable and as good as we can wish them to be in cases of normal senile cataract. The corneal wounds all healed very well and in one case even a disagreeable complication (spastic entropium of the lower lid) produced neither irritation nor suppuration of the wound. In spite of the remaining of cortical substance in the anterior chamber, no iritis developed, and judging from my cases, it does not appear to be justified, to give a bad prognosis with regard to the healing after cataract extraction in diabetic individuals. [Case I., read Jaeger No. 5, with, $+2\frac{3}{4}$ at 12″; visual acuity not given, but said to be very good with+4. Case II., R. E. Jaeger No. 2, with$+2\frac{3}{4}$ and with+4 at distance, "as well as he ever had seen;" L. E. (secondary cataract) read Jaeger No. 8 with$+2\frac{3}{4}$, visual acuity not given. Case III., R. E., with$+2\frac{3}{4}$ Jaeger No. 4, for distance selects+4, as giving him as good sight as he had in his best days; L. E., does not recognize letters of Jaeger No. 8, with$+2\frac{3}{4}$. Some opacities in pupillary area. Case IV., patient cannot read, but has good vision, of which he will, how-

ever, not allow an exact examination, in order not to lose the subsistence from his community.—EDITOR.]

I have not encountered any complications in the retina or choroid neither before nor after the operation, if I do not want to count here the capsular opacities in the third case. I think it useful to add to my observations a few words on the condition of the suspensory ligament and the hyaloid membrane. In the diabetic subjects I think they are more easily ruptured than in the normal condition. In the first case in which I could not notice any trembling of the iris the suspensory ligament must have been relaxed, because that part of the lens which corresponded with the corneal incision, became dislocated forwards and pressed the iris forwards. I do not think that the ligament was ruptured by a rapid loss of the aqueous humor, because then the vitreous body would have prolapsed and thus the replacement of the prolapsed iris [which was successfully done. EDITOR] could not have been accomplished. In the second and third cases a detachment of a part of the capsule from the suspensory ligament must have taken place and this have been moved into the pupil in consequence of the traction of the cystotome during capsulotomy. In the third case it is possible that in making the capsulotomy the hook attacked the thickened part of the capsule; this would easily explain the rupture of the suspensory ligament In cases two and four the loss of vitreous body seems to have been due to the tearing of the suspensory ligament and of the hyaloid membrane during the capsulotomy in a part which must have been quite a distance from the corneal wound; had it been near this wound its lips would have gaped.

It seems to me, that in the common senile cataract and even in cases of traumatic cataract, the suspensory ligament is generally more resistant. In diabetes these delicate tissues of the eye, perhaps, become atrophied, more brittle and less resistant. This could explain also the observation made by Hirschberg, that the pigmentary layer of the iris remained behind after an iridectomy. O. Becker does not say anything about the condition of the suspensory ligament in cases of cataract, and I believe it is not very easy to get microscopical specimens of it. My observations, undoubtedly, teach that we must be very care-

ful when operating for diabetic cataract and not force a complete expulsion of the cortical substance, when this is not easily accomplished. * * *

In an article by F. Hosch on Experimental Studies on Cysts of the Iris, published in *Virchow's Archiv*, we find the following statements:

* * * In four cases minute pieces of skin and hair (from the rabbit) were brought upon the iris and observed during from forty to forty-eight days. In one case no irritation appeared at first, but on the 24th day suppuration took place in the anterior half of the eyeball, which, however, disappeared again in a few days. The anatomical examination made on the fortieth day showed the anterior chamber and the iris filled with pus cells, the lamellæ of the cornea separated from each other by round cells. The foreign body which was nowhere adherent to the iris, consisted of a dense network of hair; in some places a network of fibres was found, having nuclei which were stained deeply by carmine. In the other cases the presence of the foreign body was well born and it led in every case to the same result, viz., to the formation of a large cyst, lined with regular pavement epithelium and filled with an atheromatous substance. This cyst lies in the iris and is attached to the cornea by a fibrous tissue containing stumps of hair and sections of glandular canals. Besides these large cysts we find in most specimens one or more smaller ones with the same lining of epithelium and the same contents.

I think the formation of these cysts cannot be better explained than by assuming them to have been formed within the transplanted piece of skin, in the manner of atheromatous cysts, *i. e.*, by the accumulation of the glandular secretion within the lumen and the orifice of a sebaceous gland. We have, therefore, here, too, to deal with retention cysts. When this process takes place within several adjacent glands, the result may, of course, be a cyst with several chambers, as they have been seen in the iris and been described by several authors.

I would like to go a step further and to give it as my opinion, that even those cysts of the iris which were undoubtedly

formed around cilia, have possibly an analogous origin. * * * * * * My opinion, therefore, is that the pearl (epidermoid) tumors of the iris as well as the so-called serous cysts of this membrane (and I cannot find a fundamental but only a difference in degree between the two) are usually caused by the fact that during the preceding traumatism parts of such tissue were thrown into the anterior chamber, which enclosed organs apt to retain their contents and secretion. •This does, of course, not deny that once in a while the cyst may originate in the manner described by von Wecker or in some other way; but these are then, probably, exceptional occurrences.